regulated by wild and mutant HBx, demonstrated a lower level in the LO2/HBx-d382 cell. Further characterization of miR-338–3p revealed that it negatively regulated cellular profileration. Cell cycle analysis showed that miR-338–3p induced cell cycle arrest at the G1/S phase. A dual-luciferase reporter assay demonstrated that the 3′UTR of CyclinD1 were directly bound to miR-338–3p and western blotting analysis further indicated that miR-338–3p down-regulated the expression of CyclinD1.
Conclusion: This study demonstrates that HBx can influence cellular miRNA expression. The deregulation of the expression of miR-338–3p by HBx may represent a potential novel pathway which HBx acts to deregulate cell proliferation leading to hepatocarcinogenesis.

PP-106 Relationship between HBsAg, HBcAg expression and serum HBV DNA level in 140 patients with chronic hepatitis B

Y. Yan[1]*, L. Mai[1], G.L. Lin[1], J. Liu[1], J.Y. Zhu[1]. [1]*The Third Affiliated Hospital of Sun Yat-sen University, China*

Objective: The aim of this study was to investigate the relationship between HBsAg, HBcAg expression and serum HBV DNA level.
Methods: The expression of HBsAg, HBcAg in the livers of 140 patients with chronic hepatitis B was detected by immunohistochemistry. And the level of serum hepatitis B virus DNA (HBV DNA) was tested. Statistical significance was assessed using One-Way analysis of variance (ANOVA).
Results: Serum HBV DNA level in 13 patients with HBsAg (−~±), 108 patients with (+~++) and 19 patients with (+++~++++) was 5.313±1.874 copies $\log_{10}$/ml, 6.010±2.016 copies $\log_{10}$/ml and 5.664±1.548 copies $\log_{10}$/ml respectively (P = 0.408). Serum HBV DNA level in 42 patients with HBsAg (−~±), 79 patients with (+~++) and 19 patients with (+++~++++) was 5.886±1.997 copies $\log_{10}$/ml, 5.968±2.020 copies $\log_{10}$/ml and 5.634±1.551 copies $\log_{10}$/ml respectively (P = 0.800).
Conclusions: The expression of HBsAg, HBcAg in the liver does not correlate with serum HBV DNA level.

PP-107 Cause analysis of chronic HBV infected patients without antiviral therapy in the Pearl River Delta region

G.L. Lin[1]*, X.J. Zhang[1], Y. Yan[1], Y.K. Wu[1], X.Y. Li[1], L.Q. Yang[1], Y.T. Chong[1]. [1]*The third affiliated hospital of Sun Yat-sen University, China*

Objectives: To analyze the causes of the patients with chronic Hepatitis B without taking antiviral therapy and the strategies of dealing with it.
Method: We make long-termed observation on the patients with chronic Hepatitis B Virus (HBV) infection, who were voluntarily to be followed up in our clinic department, and analyze statistically the objective and subjective causes of patient without taking antiviral therapy.
Results: In total eligible 951 cases, 424 cases didn't receive the antiviral therapy. 105 out of 424 cases had the indications of the antiviral therapy (105/424, 24.8%), the other 319 cases had no indications of the therapy (319/424, 75.2%). The ratio of female (124/202, 61.4%) who didn't get the antiviral therapy was significant higher than that of male (300/749, 40.0%). 49 out of 105 cases who had the indications of the antiviral therapy worried about the unhealthful effect on their fertility by the antiviral drugs and put off antiviral therapy (49/105, 46.7%); 31out of 105 cases could not pay for the antiviral therapy (31/105, 29.5%); 19 out of 105 cases queried the safety of the antiviral drugs and uncertainty of course of the treatment (19/105, 18.1%). 6 out of 105 cases were because of poor compliance (6/105, 5.7%).
Conclusions: No antiviral indications was the main cause of the untreated group. The causes of that patients with indications didn't receive antiviral therapy were that worrying about their fertility, fees of the treatment hard to bear, querying the safety of the antiviral drugs and uncertainty of course of the treatment, and poor compliance. Formal long-termed follow up by the clinicians, good communications between clinicians and patients and health education might improve the effects of anti-HBV treatment.

PP-108 Investigation on serum HBV viral loads and the changes of liver pathological features in 158 patients with chronic hepatitis B

Y. Yan[1]*, L. Mai[1], J. Liu[1], J.Y. Zhu[1], Y. Zhang[1]. [1]*The Third Affiliated Hospital of Sun Yat-sen University, China*

Objective: To investigate the relationship between serum HBV DNA loads and liver pathological changes in the patients with chronic hepatitis B.
Methods: The relationship among HBV DNA loads, live histological inflammation grades and fibrosis stages of 158 cases was analyzed.
Results: The serum HBV DNA loads in HBeAg-positive group with inflammation grades G_{0-1} (6 patients), G_2 (74 patients) and G_{3-4} (25 patients) were 5.580±1.098 copies $\log_{10}$/ml, 6.520±2.004 copies $\log_{10}$/ml and 6.950±1.467 copies $\log_{10}$/ml respectively. There was no significant difference in patients of three inflammation grades (P = 0.250). The serum HBV DNA loads in HBeAg-positive group with liver tissues fibrosis stages of S_{0-1} (23 patients), S_2 (56 patients), S_{3-4} (26 patients) were 6.599±1.832 copies $\log_{10}$/ml, 6.559±2.012 copies $\log_{10}$/ml, 6.562±1.601 copies $\log_{10}$/ml respectively, the difference was not significant (P = 0.996). The serum HBV DNA loads in HBeAg-negative group with inflammation grades G_{0-1} (8 patients), G_2 (17 patients) and G_{3-4} (28 patients) were 2.132±1.875 copies $\log_{10}$/ml, 4.745±2.250 copies $\log_{10}$/ml and 5.581±2.305 copies $\log_{10}$/ml respectively. The serum HBV DNA level in patients with G_2 and G_{3-4} inflammation grades was significant higher than in patients with G_{0-1} inflammation grades (P = 0.001). The serum HBV DNA loads in HBeAg-negative group with liver tissues fibrosis stages of S_{0-1} (10 patients), S_2 (45 patients), S_{3-4} (18 patients) were 2.689±3.225 copies $\log_{10}$/ml, 5.127±1.833 copies $\log_{10}$/ml, 5.375±2.410 copies $\log_{10}$/ml respectively. The serum HBV DNA level in patients with fibrosis stages of S_2 and S_{3-4} was significant higher than in patients with fibrosis stages of S_{0-1} ($P = 0.005$).
Conclusions: The serum HBV DNA level does not correlate with the inflammation grades and fibrosis stages of liver tissues in HBeAg-positive patients. The serum HBV DNA loads display a positive correlation with the inflammation grades and fibrosis stages of liver tissues in HBeAg-negative patients.

PP-109 Association of IL-6 gene polymorphism and its levels in HBV related hepatocellular carcinoma progression in India

R. Saxena[1], Y.K. Chawla[2], J. Kaur[1]*. [1]*Department of Biochemistry, Postgraduate Institute of Medical Education and Research, Chandigarh, 160 012, India*, [2]*Department of hepatology, Postgraduate Institute of Medical Education and Research, Chandigarh, 160 012, India*

Objectives: Hepatitis B Virus (HBV) infection is a primary risk factor for hepatocellular carcinoma (HCC), the fifth most frequent cancer, worldwide. The present study was undertaken to analyze the association of *IL-6* (–572) and

(−597) polymorphisms and IL-6 levels with hepatitis B virus related HCC risk in Indian population.
Methods: Five groups of subjects were enrolled viz. control (n = 100), HBV-carriers (n = 60), chronic active HBV (n = 60), HBV-cirrhotic (n = 60) and HBV-related HCC (n = 53). PCR-RFLP was performed to study various polymorphic forms of *IL-6* and levels in PBMCs were estimated by ELISA. Genotype distributions were compared using chi square analysis and the odds ratios (ORs) and 95% CI were calculated to express the relative risk.
Results: In *IL-6* (−*572*), the GC genotype, was in negative association ($p < 0.001$) with HCC, among controls, while it was a significant risk factor ($p < 0.001$) for the same, among HBV-carriers. In contrast, the CC genotype was a risk factor ($p < 0.001$) for progression the disease to cirrhosis among controls and HBV-carriers (OR: 3.2 and 4.0 respectively). In case of *IL-6*(−597), GA genotype significantly increased ($p < 0.001$) HCC risk, both among controls and HBV-carriers. The IL-6 levels were found to be significantly lower in all the diseased groups, with reference to controls. However, levels were significantly higher in cirrhotic group when compared with the carrier and active hepatitis group. Moreover, in both *IL-6*(−572) and (−597) heterozygotes were found to have lower IL-6 levels as compared to those having wild genotypes.
Conclusions: Polymorphic forms of *IL-6* and basal IL-6 levels share a strong association with HBV-HCC risk in Indian population and thus should be further evaluated as candidate genes to determine individual susceptibility for the same.

PP-110 Chinese herbal medicines (CHM) personalized therapy for HBeAg(+) chronic hepatitis B Chinese patient with suboptimal response to nucleoside and modeling

Y.G. Ye[1]*, L.Q. Min[2]. [1]*Traditional Chinese Internal Medicine Key Laboratory of China Education Ministry, Dongzhimen Hospital, Beijing University of Chinese Medicine, Beijing 100700, P. R. China,* [2]*Mathematics and Physics School, University of Science and Technology Beijing, Beijing, 100083, China*

Objectives: Evaluate the efficacy of Tiaoganjanpihexue (a traditional Chinease medicine term) CHM-prescription for treating a 31-year-old female HBeAg(+) chronic hepatitis B Chinese patient with suboptimal response to Adefovir Dipivoxil (AD) for 63 weeks switch to AD + lamivudine (LVD) for 18 weeks, and to AD for 45 weeks, and to Telbivudine (TEB) for 24 weeks. Modeling the dynamics of the anti-HBV infection treatments.
Methods: During 2007−2010, the patient was treated by AD, AD+LVD, AD and TEB continuously. Her baseline characteristics were HBV DNA = 3.48×10^7 cps/ml, ALT = 103.2 I/U, HBeAg = 1.400 S/CO, Anti-HBe = 0.213S/CO and those at week 150 were HBV DNA <1000 cps/ml, ALT <14 I/U (over 100 weeks), HBeAg = 3.840S/CO, Anti-HBe = 1.061S/CO. After 16 weeks' stopping treatment, her HBV DNA was returned to 6.8×10^3 and other characteristics were almost the same as those at week 150. Using the CHM Tiaoganjanpihexue (consisting of 21 ingredients) treated the patient, two times daily and decoction after meals.
Results: After 4 weeks' treatment her HBeAg and anti-HBe reduced to 0.850S/CO and 0.528S/CO. Following 21 weeks' addtional treatments, her HBeAg had been kept seroconversion and HBV DNA reduced to 1.40×10^3 cps/ml. During the 25 week's Tiaoganjanpihexue therapy, her mean ALT = 19.03 I/U. A mathematical new model is introduced to model the dynamics of the anti-HBV infection treatment. The simulated curve is in good agreement with the clinic tested patient's HBV DNA.
Conclusions: The CHM Tiaoganjanpihexue has a specific function that is able to activate patient's immune function to suppress HBV directly but not injure the patient's hepatocytes.
Acknowledgments: This work is jointly supported by the 11th 5-Year Plan Key Research Project of China (No. 2008ZX10500-006) and the NNSF of China (No.61074192).

PP-111 Hepatitis B virus (HBV) subgenotypes and mutations in core promoter and precore/core and their clinical implications in Xinjiang Uighur patients

X.F. Sun[1]*, Y.X. Zhang[1], S.J. Wen[2], J.L. Hou[2], H. Liu[1], Z.H. Wang[2]. [1]*First Affiliated Hospital, Xinjiang Medical University,* [2]*Nanfang Hospital, Nanfang Medical University, China*

Objective: To detecte HBV subtypes, mutation of core promoter and precore/core and clinical features among Xinjiang Uygur patients with chronic HBV infection.
Methods: PCR-RFLP was used to detect the subtypes, core promoter and precore/core of HBV in 109 Uygur patients with chronic HBV, and analyses of the relationship between mutation and clinical features.
Results: In 109 Uygur patients with chronic HBV group, there were 9 cases with HBV genotype B infection who were HBV Ba subtype, 50 cases with HBV genotype C and 27 cases with C1 subtype i, 23 cases with C2 subtype, 32 cases with C/D recombinant i, 18 cases with HBV genotype D infection. According to the progress of the HBV infection, G1896A mutation in HBV precore or core region had no significant difference in chronic hepatitis B and cirrhosis, but A1762T/G1764A mutation in the HBV subtype C1 and C2 and i Ba have significantly increased in cirrhosis.we fund that A1762T/G1764A mutation in HBV C/D recombinant infection is low.
Conclusion: Uygur patients with CHB have more HBV C/D recombinant, and C1 subtype infection. A1762T/G1764A mutation in the HBV subtype C1 and C2 subtypes have significantly increased.

PP-112 Genotyping of hepatitis B virus in 280 patients infected with hepatitis B Virus and its clinical significance

W.D. Li[1]*, J.L. Chen[1], C.Q. Li[1]. [1]*Beijing DiTan Hospital, Capital Medical University, China*

Objective: Previous studies showed that HBV genotype correlated with HBV transmission, clinical disease spectra, progression, prognosis, antiviral effect, etc. This study is aimed to evaluate the clinical significance of HBV genotype.
Methods: HBV genotypes were analyzed in 280 patients infected with hepatitis B virus. Liver function, HBV markers including PreS1, HBV DNA levels and T cell subsets of these patients were also measured.
Results: HBV genotyping had a geographical distribution. Genotype C was mainly prevalent in patients from north china while Genotype B from south china. In the progression of asymptomatic carrier or acute hepatitis to chronic hepatitis, liver cirrhosis and liver cancer, genotype C increased while genotype B decreased. Genotype C and genotype BC showed lower levels in prealbum (Pre-A) and album (ALB) and lower ratio of album to globulin (A/G) than genotype B ($P = 0.02$, $P = 0.03$, $P = 0.01$ and $P = 0.005$, $P = 0.001$, $P < 0.001$, respectively), but showed higher levels in globulin (GLO) than genotype B ($P < 0.001$ and $P = 0.01$, respectively). Genotype C showed lower levels in cholinesterase (CHE) than genotype B ($P = 0.007$). There